In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: May 27, 2021

ALAN ARCHER,	*	UNPUBLISHED
Petitioner,	*	No. 15-656V
v.	*	Special Master Gowen
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Entitlement; Ruling on the Record; Tetanus, diphtheria, and acellular pertussis ("Tdap"); Transverse Myelitis; Onset.
Respondent.	*	

Diana L. Stadelnikas, Maglio Christopher & Toale, Sarasota, FL, for petitioner.
Sarah C. Duncan, U.S. Department of Justice, Washington, D.C., for respondent.

DECISION ON ENTITLEMENT[1]

On June 24, 2015, Alan Archer ("petitioner") filed a petition in the National Vaccine Injury Compensation Program.[2] Petition (ECF No. 1). Petitioner alleges that as a result of receiving the tetanus, diphtheria and acellular pertussis ("Tdap") vaccine on August 7, 2012, he suffered transverse myelitis. *Id.* at ¶¶ 1-6. On March 3, 2020, petitioner filed a motion for a ruling on the record. Petitioner's ("Pet.") Motion ("Mot.") (ECF No. 76). Respondent filed a response to petitioner's motion on June 29, 2020. Respondent's ("Resp.") Response to Petitioner's Motion for a Ruling on the Record (ECF No. 89). On July 27, 2020, petitioner filed

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[1] Pursuant to the E-Government Act of 2002, *see* 44 U.S.C. § 3501 note (2012), because this decision contains a reasoned explanation for the action in this case, I am required to post it on the website of the United States Court of Federal Claims. The court's website is at http://www.uscfc.uscourts.gov/aggregator/sources/7. **This means the decision will be available to anyone with access to the Internet.** Before the decision is posted on the court's website, each party has 14 days to file a motion requesting redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). "An objecting party must provide the court with a proposed redacted version of the decision." *Id.* **If neither party files a motion for redaction within 14 days, the decision will be posted on the court's website without any changes.** *Id.*

[2] The National Vaccine Injury Compensation Program is set forth in Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 to 34 (2012) (hereinafter "Vaccine Act" or "the Act"). Hereinafter, individual section references will be to 42 U.S.C. § 300aa of the Act.

a reply to respondent's response. Pet. Reply (ECF No. 91).

After fully reviewing all of the evidence presented in this case and in accordance with the applicable legal standards, I hereby **GRANT** petitioner's motion for a ruling on the record and I find that petitioner has not established entitlement, thus his petition shall be **dismissed.**

I. Procedural History

On June 24, 2015, petitioner filed his claim alleging that the Tdap vaccine administered on August 7, 2012 caused-in-fact the development of transverse myelitis. Petition at Preamble. On September 2, 2015, I held an initial status conference, ordering petitioner to file additional medical records to verify the date the vaccine was administered. Scheduling Order (ECF No. 8). After petitioner filed updated medical records, respondent filed a status report on November 9, 2015 stating, "Based on the review of this case by the Division of Injury Compensation Programs, Department of Health and Human Services, respondent does not believe that engaging in settlement discussions with petitioner is appropriate at this time." Resp. Status Report (ECF No. 12). As such, the parties were ordered to file expert reports. *See* Scheduling Order (Nov. 9, 2015).

On October 24, 2016, petitioner filed an expert report by Dr. Agnes Jani-Acasdi[3] and supporting medical literature. Pet. Exs. 18-45 (ECF Nos. 23-47). On April 7, 2017, respondent filed an expert report by Dr. Peter D. Donofrio[4] and supporting medical literature. Resp. Exs. A-B). Respondent also filed the Rule 4(c) report the same day. Resp. Report ("Rept.") (ECF No. 31). In the Rule 4(c) report, respondent stated that "petitioner has yet to establish that he has transverse myelopathy or auto-inflammatory myelopathy…or 'longitudinally extensive TM or neuromyelitis optic spectrum disorder ("NMOSD"). Resp. Rept. at 10. Additionally, respondent also argued that petitioner's expert, Dr. Jani-Acsadi's theory is not supported by a " 'reputable' or 'reliable' scientific or medical explanation." *Id.* at 11. Respondent further argues that petitioner has not provided evidence establishing that there was a proximate temporal relationship between his Tdap vaccination and his alleged injury. *Id.* at 12. Specifically, respondent states that petitioner's expert, "does not specify when the onset of petitioner's

[3] Dr. Agnes Jani-Acsadi, M.D., was a neurologist at the University of Connecticut School of Medicine. Pet. Ex. 19 at 1. Dr. Jani-Acsadi received an undergraduate degree in 1981 and a medical degree from the University Medical School Pecs in Hungary. *Id.* While she worked at the University of Wisconsin, she cloned the gene for Duchenne muscular dystrophy. *Id.* Dr. Jani-Acsadi completed her residency training in neurology at Wayne State University, servicing as chief resident and following a neuromuscular fellowship, she was a staff neurologist at Wayne State University until 2010. *Id.* She became the Interim Chair for the Department of Neurology at UConn and Hartford Hospital. *Id.* Dr. Jani-Acsadi had published multiple articles in the field of neurology, including about chronic demyelinating polyneuropathy ("CIDP"). *Id.* at 2-3. Finally, she had a Neuromuscular Medicine Board Certification. *Id.* at 2. Unfortunately, Dr. Jani-Acsadi passed away in 2018.

[4] Dr. Peter Donofrio is a professor of neurology and Director of Neuromuscular Division at Vanderbilt University School of Medicine, and Director of the EMG lab at Vanderbilt University Medical Center. Resp. Ex. B. Dr. Donofrio received his medical degree from Ohio State University School of Medicine in 1975, and completed his internal medicine residency at Good Samaritan Hospital in Ohio. *Id.* Dr. Donofrio completed a neurology residency at the University of Michigan Medical Center and a neuromuscular fellowship at the University of Michigan. *Id.* He is board certified in internal medicine, neurology, electromyography and neuromuscular science. *Id.* Dr. Donofrio has experience in evaluating and caring for patients with neurological conditions, including transverse myelitis, Guillain'-Barre syndrome ("GBS") and CIDP, among others. *Id.*

symptoms occurred and that Dr. Jani-Ascadi notes that "there is uncertainty regarding the 'proper onset interval at the margins' such that several days to three months or longer would be an appropriate latency between vaccination and the onset of symptoms. *Id.* at 13; Pet. Ex. 18 at 6.

On October 12, 2017, I held another status conference in the case. Order (ECF No. 39). During this status conference, petitioner stated that he had retained another expert in the case, as his original expert was no longer able to continue due to a medical disability. *Id.* The parties were ordered to file supplemental expert reports. *Id.*

On March 12, 2018, petitioner filed a supplemental expert report by Dr. Matthew Imperioli[5] and supporting medical literature. Pet. Exs. 52-57. Respondent filed a supplemental expert report by Dr. Donofrio on August 9, 2018. Resp. Ex. C (ECF No. 66)[6]. Pursuant to Vaccine Rule 5, I held a status conference, indicating that the onset of petitioner's symptoms in relation to the Tdap vaccination was outside the "generally expected time frame for vaccine reactions," but that according to medical literature, "post-vaccination onset of TM could take as long as three months." Scheduling Order at 1 (ECF No. 53). I also explained that the 2-3 month period between the onset and nadir of symptoms presents another challenge with petitioner's claim. *Id.* I stated that, "This is well outside the 4 hour-21 day timeframe proposed by the TM Consortium Working Group," but observed that both petitioner and respondent's experts agreed that the "working group's diagnostic criteria may be too restrictive for clinical practice." *Id.*; *see also* Pet. Ex. 52 at 5; Resp. Ex. C at 3. Finally, I explained that petitioner's medical records from the Mayo Clinic, supported a monophasic inflammatory process, instead of a syrinx. *Id.* at 2. I explained that the medical evidence and opinion evidence appeared to be more in favor of petitioner's claim, but that any demand made by petitioner should be discounted based on the litigative risk. *Id.*

The parties engaged in unsuccessful litigative risk settlement discussions. On February 25, 2019, respondent filed a supplemental report from Dr. Donofrio and a first report from Dr. Robert Fujinami, Ph.D.[7] Resp. Exs. G-H9 (ECF Nos. 61-2). On July 18, 2019, petitioner filed a responsive report from Dr. Imperioli and supporting medical literature. Pet. Ex. 59 (ECF No. 68).

[5] Dr. Matthew Imperioli is a neurologist at the University of Connecticut School of Medicine. Pet. Ex. 53. He received his undergraduate degree from Massachusetts College of Pharmacy and Health Science and his medical degree from St. George's University in 2010. *Id.* at 1. Dr. Imperioli did his residency in neurology at the John Dempsey Hospital/Hartford Hospital. *Id.* at 2. He is board certified in neurology and neuromuscular medicine. *Id.* Dr. Imperioli is currently a professor at the University of Connecticut in the Neurology Department. *Id.*

[6] On June 14, 2019, respondent's exhibits C-F were struck for failure to properly bate stamp and refiled on June 17, 2019.

[7] Dr. Robert Fujinami is a professor in the Department of Pathology, Division of Microbiology and Immunology, and an adjunct progressor in the Department of Neurology at the University of Utah School of Medicine. Resp. Ex. I. He received his undergraduate degree from University of Utah in 1972 and his Ph.D. in Immunology-Microbiology from Northwestern University in 1977. *Id.* at 1. He did his postdoctoral training at the Scripps Research Institute. Resp. Ex. H at 1. He then went to the University of California as an Associate Professor in the Department of Pathology and investigated how viruses trigger autoimmune demyelinating disease and the immune mechanisms that lead to neuroinflammatory disease. *Id.* Dr. Fujinami investigates neuroinflammation in the context of CNS autoimmune disease and how infections can initiate seizures and epilepsy. Resp. Ex. H at 1. He has served on medical panels, including the Worth Health Organization ("WHO"), National Institutes of Medicine,

I held another status conference in the case after reviewing the newly filed expert reports on August 12, 2019. Scheduling Order (ECF No. 69). During the status conference, I explained that the medical records and statements in petitioner's affidavit reflect that onset of symptoms associated with myelopathy occurred after receipt of the Tdap vaccine on August 7, 2012. *Id.* at 2. I noted that the medical records provide different accounts for onset, but that the initial symptom was abdominal pain that occurred in September or early October 2012. *Id.* I also clarified my opinion relating to petitioner's diagnosis. *Id.* at 3. I explained that I agree with respondent's expert that petitioner's symptoms did not reach their nadir within 21 days of onset; petitioner's nadir was marked by significant gait impairment; and plateaued by December 2012 or early January 2013. *Id.* at 2. I noted that based on review of the medical records in particular the MRIs, I disagreed with Dr. Donofrio's opinion that there was no evidence of transverse myelitis in the spinal cord and that a syrinx at the C7-T1 level was the cause of petitioner's symptoms, not transverse myelitis. *Id.* at 2-3. I recommended that petitioner file a supplemental affidavit regarding his pre-vaccination medical history, if necessary, and the parties propose further proceedings in this case.

On October 11, 2019, the parties filed a joint status report stating, "After careful consideration, respondent is not interested in pursuing settlement negotiations. Respondent is amenable to a ruling on the record or an entitlement hearing; petitioner requests an entitlement hearing. Accordingly, the parties respectfully request a status conference…" Joint Status Rept. (ECF No. 73). On October 31, 2019, a status conference was held, where the parties confirmed that they were amenable to entitlement being adjudicated on the record. Scheduling Order (ECF No. 74).

II. Legal Standard

The Vaccine Act was established to compensate vaccine-related injuries and deaths. § 300aa-10(a). "Congress designed the Vaccine Program to supplement the state law civil tort system as a simple, fair and expeditious means for compensating vaccine-related injured persons. The Program was established to award 'vaccine-injured persons quickly, easily, and with certainty and generosity.'" *Rooks v. Sec'y of Health & Human Servs.,* 35 Fed. Cl. 1, 7 (1996) (quoting H.R. No. 908 at 3, *reprinted in* 1986 U.S.C.C.A.N. at 6287, 6344).

A petitioner bears the burden of establishing his or her entitlement to compensation from the Vaccine Program. The burden of proof is by a preponderance of the evidence. § 300aa-13(a)(1). A petitioner may prevail by proving either that (1) the vaccinee suffered an injury listed on the Vaccine Injury Table with onset beginning within a corresponding time period following receipt of a corresponding vaccine (a "Table Injury"), for which causation is presumed or that (2) the vaccinee suffered an injury that was actually caused by a vaccine. Under either method, however, the petitioner must also show that the vaccinee "suffered the residual effects or complications of the illness, disability, injury, or condition for more than six months after the administration of the vaccine." Section 11(c)(1)(D)(i).

Institute of Medicine-National Academic of Science, and the National Multiple Sclerosis Society. *Id.* at 2. Additionally, he has published multiple articles on molecular mimicry. *Id.*

In the present case, petitioner does not allege a Table injury. Rather, he alleges that the August 7, 2012 vaccination was the "cause-in-fact" of his development of transverse myelitis. Petition at Preamble; Pet. Mot. at 1. Thus, he bears the burden of establishing actual causation.

To prove causation-in-fact, the petitioner must "show by preponderant evidence that the vaccination brought about the injury by providing 1) a medical theory connecting the vaccination and injury; 2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and 3) a showing of proximate temporal relationship between vaccination and injury." *Althen v. Sec'y of Health & Human Servs.*, 418 F. 3d 1274, 1278 (Fed. Cir. 2005). There must be preponderant evidence for each *Althen* prong. *Caves v. Sec'y of Health & Human Servs.*, 100 Fed. Cl. 119, 132 (2011), *aff. per curiam*, 463 Fed. Appx. 932 (Fed. Cir. 2012).

The preponderance of the evidence standard requires the petitioner to demonstrate that it is "more likely than not" that the vaccine caused the injury. *Moberly v. Sec'y of Health & Human Servs.*, 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Human Servs.,* 931 F.2d 867, 873 (Fed. Cir. 1991). A petitioner must demonstrate that the vaccine was "not only [a] but for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Human Servs.,* 135 F.3d 1344, 1352-53 (Fed. Cir. 1999); *Pafford v. Sec'y of Health and Human Servs.*, 451 F.3d 1352, 1355 (Fed. Cir. 2006). Causation is determined on a case-by-case basis, with "no hard and fast *per se* scientific or medical rules." *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). A fact-finder may rely upon "circumstantial evidence" which is consistent with the "system created by Congress, in which close calls regarding causation are resolved in favor of injured claimants." *Althen*, 418 F. 3d at 1280.

If the petitioner makes a *prima facie* case supporting vaccine causation-in-fact, the burden shifts to respondent to show by a preponderance of the evidence that the injury is instead due to factors unrelated to the administration of the vaccine. *Deribeaux v. Sec'y of Health & Human Servs.*, 717 F.3d 1363, 1367 (Fed. Cir. 2013) (citing § 13(a)(1)(B)). Respondent has the burden of demonstrating that: "[A] factor unrelated to the vaccination is the more likely or principal cause of injury alleged. Such a showing establishes that the factor unrelated, not the vaccination, was 'principally responsible' for the injury. If the evidence or alternative cause is seen in equipoise, then the government has failed in its burden of persuasion and compensation must be awarded." *Knudsen*, 35 F.3d at 551.

Additionally, medical records are generally considered trustworthy. *Cucuras v. Sec'y of Health & Human Servs.*, 993 F. 2d at 1525, 1528 (Fed. Cir. 1993). Where medical records are clear, consistent and complete, they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Human Servs.*, No. 03-1585V, 2005 WL 6117475 (Fed. Cl. Spec. Mstr., Dec. 12, 2005). Medical records may be outweighed by testimony that is given later in time that is "consistent, clear, cogent, and compelling." *Camery v. Sec'y of Health & Human Servs.,* 42 Fed. Cl. at 381, 391 (1998).

If there is a dispute as to the nature of the petitioner's injury, the special master may opine on the nature of the petitioner's injury." *Contreras v. Sec'y of Health & Human Servs.,*

844 F. 3d 1363, 1368 (Fed. Cir. 2017) (citing *Hibbard v. Sec'y of Health & Human Servs.,* 686 F. 3d 1355 (Fed. Cir. 2012); see also *Broekelschen v. Sec'y of Health & Human Servs.,* 618, F.3d 1339 at 1346 (Fed. Cir. 2010). In *Broeklschen,* the Federal Circuit stated that it was appropriate for the special master to first determine which injury is best supported by the evidence presented in the record before applying the *Althen* test. *Broekelschen* at 1346.

III. Summary of Relevant Facts

A. Medical Records

Petitioner was 55-years old at the time when he received the Tdap vaccination on August 7, 2012. Pet. Ex. 1 at 1; Pet. Ex. 10 at 7. He had an appointment with Dr. Steven Stiles at St. Luke's Internal Medicine in Kansas City, Missouri. Pet. Ex. 10 at 7. Dr. Stiles noted existing problems of "hyperlipidemia and Type 1 diabetes mellitus." *Id.* Petitioner's physical exam was normal, and he received the Tdap vaccination. Pet. Ex. 1 at 1.

On October 18, 2012, petitioner presented to KU West Urgent Care for urinary pain and urinary frequency. Pet. Ex. 6 at 3. Petitioner reported he had urinary pain for five days and increased dramatically that day. *Id.* Petitioner informed Dr. Gary Parkhurst that he had been experiencing urinary hesitancy for several months, but "over the last several days he also begun experiencing some mild discomfort in the lower abdominal/pelvic area with slight dysuria." *Id.* at 6. Additionally, he was positive for "abdominal pain" and "decreased urine volume and difficulty urinating." *Id.* Dr. Parkhurst noted that petitioner had a slightly enlarged prostate. *Id.*

On October 23, 2012, petitioner returned to Dr. Stiles with "a week to 10-day history of upper abdominal discomfort." Pet. Ex. 10 at 5. Dr. Stiles noted petitioner, "Has a dull discomfort across the upper abdomen and occasionally gets a sharper pain in a focal area in the right upper lateral abdomen." *Id.* Dr. Stiles also wrote, "His urinary symptoms have become gradually worse. Having some nocturia issues and also has a delay in bladder emptying." *Id.* Dr. Stiles diagnosed petitioner with "abdominal pain" and "benign prostatic hypertrophy" and opined, "Abdominal pain-positional nature of symptoms highly suggestive of underlying thoracic radiculopathy or thoracic spinal stenosis. Lower urinary tract symptoms." *Id.* at 6. Dr. Stiles ordered an MRI of the thoracic spine. *Id.*

On October 26, 2012, petitioner had an MRI of his thoracic spine. Pet. Ex. 10 at 42. On the MRI report, it stated, "Indication: mid-abdominal pain and tightness. Difficulty walking. Tingling in bilateral upper extremities." The MRI of petitioner's thoracic spine showed an, "Incompletely characterized slight expansion of the *cervical and thoracic* spine to the level of T3 with central T2 hyperintensity." [8] *Id.* at 433. The radiologist wrote, "There is a wide differential for this appearance. Given central cord hyperintensity, ischemic/inflammatory process as well as an underlying malignancy cannot be excluded. Recommend further evaluation with contrast MRI of the brain, cervical spine and thoracic spine to better differentiate with these entities." *Id.* Further, he noted, "Incompletely characterized left adrenal lesion measuring up to 1 cm." *Id.* The record also states, "After talking to Dr. Stiles, the patient's symptoms are relatively mild and

[8] This MRI report did not define the level of the cervical spine at which the expansion began and did not note the subsequently seen small cyst at T1.

not acute on onset. As a result, a syrinx[9] is suspected and non-contrast and contrast [MRI] c-spine and post-contrast only T-spine MRI is recommended for further characterization." *Id.*

On October 30, 2012, petitioner had an appointment with neurologist, Dr. Steven Arkin. Pet. Ex. 3 at 20. Petitioner was evaluated for "subacute onset and progression of circumferential paresthesias and dysesthesias in the thoracic to abdominal region over the past two to three months or so." *Id.* at 20-21. Dr. Arkin wrote that, "[Petitioner]] was in his usual state of health until about two or three months ago when he started having a sense of pressure tightness in the abdomen. Since that time, he has had occasional burning sensation…On four or five occasions, he has difficulty walking if he has been in the recliner for a while…He noticed that his legs have been a bit jumpier than they have been before. He has a patch of numbness around the right knee as well as the left thigh. There can be some tingling in the same distributions. He also has some tingling in his fingers and toes at the same time." *Id.* at 21. The physical exam showed petitioner's reflexes were brisk, 3/3 at the knees and 2+/2+ at the ankles. *Id.* Petitioner had a mild ataxic gait and patches of decreased sensation in the mid-portion of the right calf, normal on the left side and decreased sensation in the abdomen around T3 to T10. *Id.* at 21. Dr. Arkin opined that petitioner "had evidence of cervical thoracic myelopathy in conjunction with slightly expanded spinal cord with what appears to be centrally located T2 hyperintensities extending from the lower cervical cord down into the thoracic region….I suspect that this will turn out to be a syrinx but the other possibilities are inflammatory/autoimmune or demyelinating/multiple sclerosis." *Id.* at 22.

Petitioner had additional MRIs of his brain and cervical and thoracic spine on November 2, 2012. Pet. Ex. 10 at 37. The MRI of petitioner's cervical spine demonstrated the "central T2 hyperintensity extending from the C6-C7 level to the T3-T4 level which is not significantly changed compared to the prior exam. There is a focal rounded T2 hyperintense lesion in the left lateral aspect of the cord at the T1 level measuring 5mm craniocaudal and 3 mm transverse which was not definitely seen on the prior exam….The cord is mildly expanded at these levels." *Id.* at 38. The radiologist's impression was, "Intramedullary central T2 signal extending from the C6-C7 to T3-T4 level as well as a left lateral cystic appearing lesion at the T1 level. Primary differential considerations include a syringomyelia, sequelae of prior inflammation (transverse myelitis)." *Id.* at 38.

Petitioner had a follow-up appointment with Dr. Arkin on November 6, 2012. Pet. Ex. 10 at 22. Dr. Arkin indicated that petitioner was being evaluated for "subacute and progressive onset of circumferential paresthesia and dysesthesias involving the abdomen associated with T2 lesion in the lower cervical and upper thoracic spinal cord. Pet. Ex. 3 at 18. Dr. Arkin also noted that petitioner had intermittent difficulty walking after being in a recliner for an extended period of time, urinary hesitancy and was hyper-reflexive in the lower extremities. *Id.* at 18. Dr. Arkin observed that the second MRI "again revealed the T2 hyperintensity…in the spinal cord between C6-C7 and T3-4." *Id.* He also noted a "…tiny 5x3mm T2 lesion just laterally within

[9] A "syringomyelia" is a slowly progressive syndrome of cavitation in the central segments of the spinal cord, generally in the cervical region, but sometimes extending up into the medulla oblongata or down into the thoracic region. It results in neurologic deficits, usually segmental muscular weakness and atrophy with a dissociated sensory loss (loss of pain and temperature sensation, with preservation of the sense of touch). *Dorland's Illustrated Medical Dictionary* 33rd ed. (2020) (hereinafter "*Dorland's"*) at 1828-29.

the cord that looks cystic." *Id.* Dr. Arkin opined, "At this point, the situation most likely represents a syringomyelia involving the spinal cord between C6-7 and T3-4. His symptoms do match up as well as hyperreflexia and some bowel and bladder dysfunction….One consideration might be a neurosurgical evaluation if his situation worsens particularly if he starts developing weakness or gait instability." *Id.* Dr. Arkin recommended petitioner undergo a lumbar puncture to "look for any evidence of [an] inflammatory situation." *Id.* He wrote, "This could be transverse myelitis associated with collagen vascular disease or other type of autoimmune pathology." *Id.* Petitioner had a lumbar puncture performed on November 26, 2012, which noted that petitioner had 8 white blood cells (WBC), which was noted as "high" and a protein CSF of 67, also noted as "high." Pet. Ex. 11 at 186.

On November 28, 2012, petitioner had an evaluation with neurosurgeon Dr. Darren Lovick. Pet. Ex. 5 at 3. Dr. Lovick wrote that, "[Petitioner] has had burning and tingling and tightness around the rib cage over the last few months now with difficulty with numbness in his legs and some weakness in his feet and difficulty with his gait. He also has noticed some troubles with urination." *Id.* After performing a physical examination and reviewing petitioner's MRIs, Dr. Lovick stated, "He is definitely myelopathic and it fits with his level of function." Dr. Lovick recommended a spinal arteriogram "in the case this could be a dural AV fistula." *Id.* at 4.

The petitioner underwent a spinal and cervical IR angiogram on December 19, 2012. Pet. Ex. 11 at 160. The results were normal. *Id.* at 163. Petitioner had a follow-up appointment with Dr. Lovick on December 26, 2012. Pet. Ex. 5 at 5. Dr. Lovick noted that petitioner did not have a dural AV fistula or any type of arteriovenous malformation. *Id.* Dr. Lovick explained to petitioner that "the possible etiologies" of his symptoms, included, "inflammatory conditions like transverse myelitis, sarcoidosis, and a demyelinating process." *Id.* He ordered petitioner begin a trial of prednisone beginning at 60 mg per day. *Id.*

On January 10, 2013, petitioner returned to Dr. Arkin. Pet. Ex. 3 at 13. Dr. Arkin repeated petitioner's medical history, noting petitioner had a normal spinal angiogram. *Id.* Petitioner had reported slight improvement, but then worsened on the prednisone taper. *Id.* The differential diagnosis continued to be "fistula versus sarcoidosis versus transverse myelitis versus neoplasia." *Id.* Dr. Arkin recommended a repeat lumbar puncture and prescribed Neurontin at 200 mg. *Id.* The repeat lumbar puncture performed on January 21, 2013 showed elevated protein in the CSF and mildly elevated pleocytosis. Pet. Ex. 3 at 4. Interestingly, Dr. Arkin continued to note fistula despite the normal arteriogram and did not mention a syrinx at this point.

Petitioner had an appointment with Dr. Arkin on January 30, 2013. Pet. Ex. 3 at 11. At this appointment Dr. Arkin noted petitioner had slightly decreased strength in his lower extremities with increased reflexes and upgoing toes. *Id.* He observed petitioner "continues to have decreased sensation, although not to the extent that he has had before." *Id.* Dr. Arkin stated, "This remains a very perplexing situation. This could be still be syringomyelia fairly stable. This could still be an occult AVM or neoplasia." *Id.* Dr. Arkin stated that he would help facilitate a second opinion at another facility and recommended a repeat MRI in the future. *Id.*

On March 18, 2013, petitioner sought a tertiary care evaluation of his condition at the Mayo Clinic and had an appointment with Dr. Joseph Y. Matsumoto for a neurology evaluation.

Pet. Ex. 7 at 17. Petitioner explained that in September 2012 he began to feel a tightness in his abdomen that was recorded as "periumbilical." *Id.* Petitioner further explained he also experienced a "sense of burning." *Id.* Dr. Matsumoto reviewed the MRI reports and noted evidence of central hyperintensity of T2 signal and the results from the lumbar punctures and noted elevated protein in the CSF from both samples. *Id.* Petitioner also reported that he was getting progressively weaker and dragging his feet and falling. *Id.* Petitioner stated that he had numbness from his knees to his feet, but that the numbness would sometimes present more proximally. *Id.* After performing a neurological exam, Dr. Matsumoto wrote,

> [Petitioner] has clear evidence of a predominately thoracic myelopathy at this time. He has weakness in an upper motor neuron distribution bilaterally. He has vibratory and joint position loss at the toes. The vibratory sensation loss extends to the knees. He has clear pinprick sensation loss, worse on the left than the right, that extends all the way up to approximately the T7 level….Reflexes are increased in the legs, and plantar reflexes are extensor. He is walking with dragging both feet and unsteadiness on tandem gait.

Pet. Ex. 7 at 18. Dr. Matsumoto diagnosed petitioner with "Myelopathy, predominately thoracic with some question of cervical myelopathy as well. The cause is uncertain." *Id.* at 18. Dr. Matsumoto also noted that a sarcoid is unlikely given the absent response to steroids. *Id.* Dr. Matsumoto ordered repeat MRIs and requested the films from the spinal angiogram. *Id.*

The same day, March 18, 2013, petitioner had additional MRIs of his cervical and thoracic spine. Pet. Ex. 7 at 31. The results showed a T2 hyperintensity centrally in the spinal cord from the C6-C7 interspace to the mid T3 level. *Id.* It also showed a focal 1-2 millimeter cystic "appearing component" at the C7-T1 interface, in the left side of the cord. *Id.* It was noted that there was no enhancement within or adjacent to the lesion. *Id.* The radiologist stated that the lesion was not consistent with a dural AV fistula, but it could represent a demyelinating process such as neuromyelitis optica or post viral myelitis. *Id.* The radiologist stated, "the absence of enhancement would be atypical for a neoplastic process or sarcoidosis." *Id.*

On March 20, 2013, petitioner had a follow-up appointment with Dr. Matsumoto following the MRIs. Pet. Ex. 7 at 14. Dr. Matsumoto ruled out evidence of AVM or AV fistula. He stated that the MRIs from October 26, 2012 looks "a bit worse than the one we have today." *Id.* Dr. Matsumoto wrote, "The area of T2 signal abnormality looks a bit wider within the cord than what we see today. Its extent looks about the same, although the films do not go to the rostral extent of the lesion." *Id.* He sent the MRIs for another review to determine if there had been any interval progression of the lesion and also referred petitioner to the Mayo Clinic's demyelinating disease experts. *Id.* After reviewing the results, Dr. Matsumoto questioned the petitioner again about his disease progression and noted petitioner stated he had gotten worse after plateauing in January. *Id.*

Another radiologist at the Mayo Clinic, Dr. David DeLone, reviewed all of petitioner's MRIs, including the MRIs from October 26 and November 2, 2012. Pet. Ex. 7 at 31. Dr. DeLone noted that the upper thoracic cord central T2 hyperintensity has not changed since October 26, 2012. He also wrote that the "small cystic appearing focus now present at C7-T1 was not clearly present on 10/26/2012." *Id.*

Petitioner met with Dr. Mark Keegan, a demyelination expert at the Mayo Clinic on March 21, 2013. Pet. Ex. 7 at 8-11. Petitioner reported that he was well until August 2012 and recalled receiving the Tdap vaccine around the same time. *Id.* at 8. Petitioner indicated that in September or earlier, he developed a "numb sensation and a tightness and burning sensation in his abdomen that was positional in nature." *Id.* Petitioner explained that in mid-October he could walk three miles with only abdominal discomfort, however, later he developed gait impairment that plateaued between December and January. *Id.* Dr. Keegan noted that the neuroimaging shows a longitudinally extensive area of abnormal signal centrally within the spinal cord. *Id.* After a physical exam, Dr. Keegan stated that, "It seems likely that [petitioner] had an inflammatory cause for the myelopathy….There was no compressive impairment. It is not clear, as was the concern, that there has been a definitive progression over time, and it does appear that it is confirmed that it has plateaued." *Id.* at 11. Dr. Keegan explained that repeat corticosteroids or plasma exchange for symptom resolution would not be beneficial at that time, but if his tests came back positive for an autoimmune condition like neuromyelitis, then he would recommend immunosuppressive medications. *Id.* Additionally, Dr. Keegan indicated that he would treat petitioner's condition as a monophasic process if petitioner's serological evaluations came back negative and would only recommend chronic immunosuppressive medications if a definite recurrent inflammatory myelopathy process was found. *Id.* Dr. Keegan recommended petitioner obtain a gait aid and pursue physical therapy. *Id.* Dr. Keegan diagnosed petitioner with "myelopathy, probable inflammatory cause." *Id.*

Petitioner met with Dr. Matsumoto later the same day. Pet. Ex. 7 at 7. Dr. Matsumoto noted that Dr. Keegan "felt the petitioner had an inflammatory myelopathy," but was waiting for serological results to determine next treatment steps. *Id.* Dr. Matsumoto explained, "If all serological evaluations come back negative, [Dr. Keegan] would recommend conservative therapy, considering this is a monophasic illness such as transverse myelitis." *Id.* Petitioner's autoimmune panel returned normal, including the NMO antibody. *Id.* at 3-4.

On March 28, 2013, petitioner had an evaluation by Dr. Brad Steinle at Physical Medicine and Rehabilitation. Pet. Ex. 8 at 3. Petitioner reported progressive difficulties with his legs since September 2012, erectile dysfunction for two years and urinary hesitancy since June 2012. *Id.* Dr. Steinle noted an enhancing lesion around C4-T6 was found and petitioner underwent extensive work-up and it "appears to be transverse myelitis." *Id.* During the physical exam, Dr. Steinle observed that petitioner had "profound proprioceptive loss in the lower limbs," and his "sensation is also diminished in a stocking glove fashion in his feet bilaterally." *Id.* at 4. Dr. Steinle assessed petitioner with "transverse myelitis with paraparesis, neuropathic pain, neurogenic bladder and bowel." *Id.* He recommended petitioner increase the dosage of gabapentin and prescribed Nuvigil, as well as, physical therapy. *Id.*

Petitioner a physical therapy evaluation on April 3, 2013. Pet. Ex. 11 at 38. The patient's medical history ("PMH") notes that petitioner was diagnosed with transverse myelitis. *Id.* During the physical examination, petitioner demonstrated weakness in his lower extremities bilaterally and decreased sensation in his lower extremities as well. *Id.* at 39. On May 1, 2013 petitioner was discharged from physical therapy. *Id.* at 45-46.

On May 2, 2013, petitioner had a follow-up appointment with Dr. Steinle. Pet. Ex. 8 at 2. Petitioner reported that physical therapy helped, and he was compliant with the home exercise program. *Id.* Dr. Steinle noted petitioner still had proprioceptive loss in the lower limbs, but he has a steadier and brisk gait with the use of forearm crutches. *Id.* Dr. Steinle again diagnosed petitioner with transverse myelitis with paraparesis, neuropathic pain and neurogenic bladder and bowel, functionally improved. *Id.*

On August 15, 2013, petitioner had an appointment with Dr. Steven Stiles for his annual physical. Pet. Ex. 10 at 2. Dr. Stiles noted that petitioner had gone to the Mayo Clinic and wrote, "This diagnosis available at this point for his neurologic issues is transverse myelitis." *Id.* During the physical exam, petitioner demonstrated mild weakness in the lower extremities and his gait was observed as mildly to moderately ataxic. *Id.* at 3.

On September 26, 2013, petitioner presented to Dr. Jennifer Elliott with the main complaint of abdominal pain. Pet. Ex. 11 at 22. Petitioner reported chronic abdominal pain that is predominantly in the right upper quadrant which feels like a "knot type sensation at times." *Id.* Petitioner described it as a "burning and tightness associated with position," that would go away when he was laying down. *Id.* Petitioner rated his pain as a 5-8 out of 10 in seating or standing positions, but a 0/10 when laying down. *Id.* During the physical exam, petitioner demonstrated intact sensation to light touch and "abnormal sensation" to pinprick in the bilateral lower extremities. *Id.* at 23. His reflexes were 2+ bilaterally in the lower extremities. *Id.* Dr. Elliott diagnosed petitioner with neuropathic abdominal pain secondary to transverse myelitis, diabetes, and low back pain. *Id.*

On November 19, 2015, petitioner had an appointment with Dr. James H. McDonald for an annual wellness exam. Pet. Ex. 50 at 17. Petitioner reported he was experiencing mild symptoms of neuropathy which was recorded as "stable." *Id.* at 18. Petitioner declined the annual flu shot at this appointment, explaining that he had transverse myelitis from the Tdap vaccine he received in 2012. *Id.* at 20. Petitioner stated that, "About 5 weeks after the vaccine symptoms started. They have remained the same. Symptoms have not gotten better or worse." *Id.* Petitioner continued to be treated by Dr. McDonald for managing diabetes and cholesterol. *See* Pet. Ex. 50; Pet. Ex. 71.

On February 21, 2017, petitioner had a neurology consult with Dr. Michael Schwartzman. Pet. Ex. 50 at 75. Petitioner was referred by his primary care physician, Dr. James McDonald. *Id.* Petitioner reported intermittent prickly dysesthesias in his arms and sometimes radiating to his upper chest. *Id.* Dr. Schwartzman reviewed petitioner's past medical history, noting a history of thoracic transverse myelitis beginning in September 2012 associated with spastic paraparesis and neuropathic pain. *Id.* Dr. Schwartzman reviewed petitioner's October 2012 MRI and observed that the differential diagnoses included syringohydromyelia, sequelae of prior inflammation or less likely a low-grade glial tumor. *Id.* at 76. During the neurological evaluation, Dr. Schwartzman observed petitioner had "superimposed decreased pinprick and temperature discrimination in the distal lower extremities with distal proximity gradient to below the knees." *Id.* at 79. Petitioner's proprioception was normal. *Id.* Dr. Schwartzman's impression was "history of idiopathic transverse myelitis; probable diabetic peripheral neuropathy of bilateral lower extremities; probable cubital tunnel syndrome; and

upper extremity dysesthesias extending into this arms and anterior chest of unclear etiology." *Id.* An EMG/NCS and repeat MRI of the cervical and thoracic spine with and without contrast was ordered. *Id.*

The EMG/NCS tests performed on February 23, 2017 showed evidence of bilateral ulnar motor neuropathies at the elbows and chronic motor axonal loss without evidence of chronic reinnervation or denervation. Pet. Ex. 50 at 82. The MRI of petitioner's cervical spine taken on March 4, 2017 noted the T2 hyperintense signal involving the cervical thoracic cord from C7-T2 with a focal 5 mm syrinx in the left aspect of the cord at the level of T1. Pet. Ex. 51 at 21. The radiologist's impression was, "Persistent abnormal cord signal at the cervicothoracic junction with overall decrease T2 signal compared to the 2012 examination, without enhancement. This suggests remote demyelinating or remote infectious/inflammatory process." *Id.*

Petitioner had a follow-up visit with Dr. Schwartzman on March 14, 2017. Pet. Ex. 51 at 8. Petitioner reported prickly dysesthesias in his chest and hands, along with intermittent numbness involving the bilateral fourth and fifth digits. *Id.* Dr. Schwartzman stated that he reviewed the Mayo Clinic's assessment and his impression was, "It was felt that [petitioner] had idiopathic transverse myelitis." *Id.* After a physical exam, Dr. Schwartzman's impression was, "History of idiopathic transverse myelitis. I suspect that his sensory symptoms are reemergence of his prior transverse myelitis; probable diabetic neuropathy; and bilateral cubital tunnel syndrome." *Id.* at 10. Petitioner was prescribed a trial of Cymbalta and referred to a neurosurgeon for potential ulnar nerve transposition. *Id.*

Petitioner underwent bilateral ulnar decompression in the fall of 2017. *See* Pet. Ex. 51 at 11-16. Petitioner reported that his chronic pain was completely resolved, and numbness significantly decreased in his upper extremities. *Id.* at 16.

On November 28, 2017, petitioner had an annual wellness exam and routine diabetic maintenance visit with Dr. McDonald. Pet. Ex. 71 at 24. It was noted that petitioner's diabetes and dyslipidemia were stable, and he was exercising at least thirty minutes a day a few days a week. *Id.* It was recorded that he had a normal gait and was able to stand without difficulty at this appointment. *Id.* at 29. He was diagnosed with controlled diabetes mellitus with neuropathy. *Id.* at 30. Petitioner continued to have medical appointments focused on his diabetic maintenance. *See* Pet. Ex. 71 at 36-54. Transverse myelitis was noted in petitioner's past medical history. *Id.*

B. Petitioner's Affidavit

Petitioner submitted a supplemental affidavit on December 12, 2017. Pet. Ex. 49. In this affidavit, petitioner stated that prior to the administration of the Tdap vaccine on August 7, 2012, he had full mobility with reasonable fortitude and could walk eighteen holes of golf. Pet. Ex. 49 at ¶ 1. He stated that he was in same condition until "late September to early October," when he began to notice minor pain near his bladder. *Id.* at ¶ 2. Petitioner also wrote that he was having trouble urinating and abdominal pain. *Id.* He described that the pain was located mostly on his right side and "the pain was like a band, with pressure across my abdomen." *Id.* The pain had built up so much, that by October 18, 2012, he sought medical assistance. *Id.*

Petitioner stated that after a second medical appointment on October 23, 2012, he continued to have pain but also experienced weakness and difficulty walking any significant distance. *Id.* at ¶ 3. Additionally, petitioner stated that he was unable to urinate and felt a sense of urgency without being able to empty his bladder. *Id.* Eventually his primary care physician arranged an MRI and an appointment with Dr. Arkin. *Id.*

Petitioner indicated that by the end of October he found it increasingly difficult to walk and the sensation in his legs were diminished. *Id.* at ¶ 4. Petitioner stated that he lost some sense of his balance which contributed to further issues with walking. *Id.*

Petitioner wrote that on the day of the vaccination, he had been seeking treatment for an enlarged prostate. *Id.* at ¶ 5. He stated that his urinary symptoms became significantly worse after vaccination. *Id.* Petitioner clarified that the progression of symptoms after the vaccination were abdominal tightness and banding sensation, pain, constipation and bladder urgency, which he stated, "began in late September." *Id.* Petitioner explained that the pain was what initially caused him to seek assistance at the Acute Care Center, then his primary care physician, which resulted in an MRI all in the month of October. *Id.* He stated that his symptoms of decreased sensation in his legs, loss of balance, pain and urinary disruption have continued since October 2012. *Id.* at ¶ 6. He continues to take Gabapentin and Tramadol daily, in addition to using a TENS unit. *Id.* at ¶ 7.

IV. Consideration of Petitioner's Diagnosis

In this case, the parties dispute whether petitioner has transverse myelitis. Pet. Mot. at 17-19; Resp. Response at 17. Petitioner's experts, Drs. Jani-Acsadi and Imperioli contended that petitioner suffered monophasic transverse myelitis following the receipt of the Tdap vaccine. Pet. Ex. 18 at 8; Pet. Ex. 52 at 2. Respondent's expert, Dr. Donofrio, contended that petitioner did not have transverse myelitis and petitioner most likely had a syrinx, which was the cause petitioner's symptoms. Resp. Ex. A at 11; Resp. Ex. C at 7. Accordingly, I find it appropriate for first determine which injury is best supported by the evidence presented.

If the existence and nature of the [petitioner's] injury itself is in dispute, it is the special master's duty to first determine which injury was best supported by the evidence presented in the record before applying the *Althen* test to determine causation of that injury. *Lombardi v. Sec'y of Health & Human Servs.*, 656 F.3d 1343, 1352 (Fed. Cir. 2011) (citing *Broekelschen v. Sec'y of Health & Human Servs.*, 618 F.3d 1339, 1346).

The Vaccine Act provides that Special Masters are to consider any medical diagnosis contained in the record. §300aa-13(b)(1). A special master must weigh and evaluate opposing expert opinions, medical and scientific evidence and the evidentiary record in deciding whether petitioners' have met their burden of proof. *Id.* The function of a special master is not to 'diagnose' vaccine-related injuries, but instead to determine based on the evidence as a whole and the totality of the case, whether it has been shown by a preponderance of the evidence that a vaccine caused the petitioner's injury. *Lombardi v. Sec. of Health & Human Servs.*, 656 F.3d 1343, 1351 (citing *Andreu*, 569 F.3d at 1382) (Fed. Cir. 2011). In *Capizzano,* the Federal Circuit

provided additional guidance as to how special masters should weigh evidence, placing emphasis on the statements of treating doctors. 440 F.3d at 1320 (Fed. Cir. 2006). The Federal Circuit stated, "*Althen III* explained that medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect' shows that the vaccination was the reason for the injury.'" *Capizzano* at 1326 (quoting *Althen,* F. 3d at 1280).

Additionally, medical records are generally considered trustworthy. *Cucuras v. Sec'y of Health & Human Servs.*, 993 F. 2d at 1525, 1528 (Fed. Cir. 1993). Where medical records are clear, consistent and complete, they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Human Servs.*, No. 03-1585V, 2005 WL 6117475 (Fed. Cl. Spec. Mstr., Dec. 12, 2005). Medical records may be outweighed by testimony that is given later in time that is "consistent, clear, cogent, and compelling." *Camery v. Sec'y of Health & Human Servs.,* 42 Fed. Cl. at 381, 391 (1998).

A. Transverse Myelitis

Inflammatory diseases of the spinal cord are collectively described as myelitis and can either be infectious or immune mediated (autoimmune). Pet. Ex. 23 at 6.[10] According to the article by Frohman et al., "Transverse myelitis syndrome may arise from various causes, but it most often occurs as an autoimmune phenomenon after infection or vaccination (accounting for 60% of cases in children)…" Pet. Ex. 64 at 1. [11] The same article states that, "Estimates of the annual incidence of idiopathic or postinfectious transverse myelitis range from 1.3 to 8 cases per million." *Id.* at 1.

Petitioner's expert, Dr. Jani Acsadi explained that transverse myelitis "is a rare clinical syndrome in which an immune-mediated process causes neural injury to the spinal cord, resulting in varying degrees of weakness, sensory alterations and autonomic dysfunction." Pet. Ex. 18 at 3. Respondent's expert, Dr. Donofrio agreed with Dr. Jani-Acsadi's description of transverse myelitis, adding that transverse myelitis, "may arise from many causes but most occur as an autoimmune phenomena after an infection, an underlying systemic autoimmune disease, or an acquired demyelinating disease such as the first presentation of multiple sclerosis or spectrum disorders of central nervous system demyelinating disease." Resp. Ex. A at 6.

The article by Karussis and Petrou explained that, "Transverse myelitis is characterized by inflammation and demyelination across both sides of one level, or segment, of the spinal cord resulting in symptoms of neurological disconnection and dysfunction below the level of the demyelinating area." Pet. Ex. 23 at 6. The article by the Transverse Myelitis Consortium Working Group ("TMCWG"), submitted by the respondent, explained that transverse myelitis is characterized "clinically by acutely or subacutely developing symptoms and signs of neurologic dysfunction in motor, sensory, and autonomic nerves and nerve tracts of the spinal cord." Resp.

[10] Karussis, D. & Petrou, P., *The spectrum of post-vaccination inflammatory CNS demyelinating syndromes,* Autoimmune Review (2013). [Pet. Ex. 23]

[11] Frohman, E. & Wingerchuk, D., *Transverse Myelitis,* 363 N. Engl. J. Med. 564-72 (2010). [Pet. Ex. 64].

Ex. A Tab 1 at 1. [12] Further, the TMCWG explained that there is "often a clearly defined rostral border of sensory dysfunction and spinal MRI and lumbar puncture often show evidence of acute inflammation." *Id.*

An article by Goh et al., regarding MRI imaging in transverse myelitis, submitted by petitioner, stated that idiopathic transverse myelitis is a diagnosis of exclusion. Pet. Ex. 63 at 1.[13] The article also explained the distinction between "longitudinally extensive transverse myelitis" and "acute partial transverse myelitis." *Id.* at 2. The article states that longitudinally extensive transverse myelitis" extends over more than three segments and involves all or most of the cross-section of the cord and "acute partial transverse myelitis" has less than two segments of eccentric or asymmetric cord involvement. *Id.* Further, this article stated, "The typical MRI appearance in transverse myelitis is a central T2 hyperintense spinal cord lesion extending over more than two segments, involving more than two-thirds of the cross-sectional area of the cord. Although any cord level can be affected, the classic description includes a preference for the thoracic cord." *Id.* Finally, this article states that enhancement is present in only 37-74% of cases. *Id.*

Both parties submitted articles that explained the Transverse Myelitis Consortium Working Group's ("TMCWG") inclusion criteria for diagnosis of transverse myelitis (idiopathic or disease associated). *See* Pet. Ex. 63; Resp. Ex. A. The TMCWG proposed the following diagnostic criteria for transverse myelitis:

Table 1 *Criteria for idiopathic acute transverse myelitis*

Inclusion criteria	Exclusion criteria
Development of sensory, motor, or autonomic dysfunction attributable to the spinal cord	History of previous radiation to the spine within the last 10 y
Bilateral signs and/or symptoms (though not necessarily symmetric)	Clear arterial distribution clinical deficit consistent with thrombosis of the anterior spinal artery
Clearly defined sensory level	Abnormal flow voids on the surface of the spinal cord c/w AVM
Exclusion of extra-axial compressive etiology by neuroimaging (MRI or myelography; CT of spine not adequate)	Serologic or clinical evidence of connective tissue disease (sarcoidosis, Behçet's disease, Sjögren's syndrome, SLE, mixed connective tissue disorder, etc.)*
Inflammation within the spinal cord demonstrated by CSF pleocytosis *or* elevated IgG index *or* gadolinium enhancement. If none of the inflammatory criteria is met at symptom onset, repeat MRI and lumbar puncture evaluation between 2 and 7 d following symptom onset meet criteria	CNS manifestations of syphilis, Lyme disease, HIV, HTLV-1, *Mycoplasma*, other viral infection (e.g. HSV-1, HSV-2, VZV, EBV, CMV, HHV-6, enteroviruses)*
Progression to nadir between 4 h and 21 d following the onset of symptoms (if patient awakens with symptoms, symptoms must become more pronounced from point of awakening)	Brain MRI abnormalities suggestive of MS*
	History of clinically apparent optic neuritis*

*Do not exclude disease-associated acute transverse myelitis.

AVM = arteriovenous malformation; SLE = systemic lupus erythematosus; HTLV-1 = human T-cell lymphotropic virus-1; HSV = herpes simplex virus; VZV = varicella zoster virus; EBV = Epstein–Barr virus; CMV = cytomegalovirus; HHV = human herpes virus.

Resp. Ex. A Tab 1 at 2. Further, the TMCWG states that to be diagnosed with idiopathic acute transverse myelitis "all of the inclusion criteria should be met and none of the exclusion criteria are fulfilled." *Id.* The same article identifies limitations to the proposed criteria. *Id.* at 3. The article explains that the exclusion of cases based on the "interval between symptom onset and

[12] Transverse Myelitis Consortium Working Group, *Proposed diagnostic criteria and nosology of acute transverse myelitis*, 59 Neurol. (2002). [Resp. Ex. A Tab 1].

[13] Christine Goh et al., *MRI in Transverse Myelitis*, 40 Journal of Magnetic Resonance Imaging, 1267-1279 (2014). [Pet. Ex. 63].

maximal deficit is arbitrary….[the authors] remain committed to distinguishing ATM from a rapidly evolving vascular myelopathy (< 4-hour progression), a slowly progressive or stuttering hereditary myelopathy, spinal cord tumor, myelopathy due to a dural arteriovenous fistula and a chronic progressive form of MS (all longer than 21 days of progression)." *Id.* Further, the authors explained that the proposed criteria represent a useful framework for future studies but will need to be prospectively validated to determine whether they appropriately categorize individuals and whether the criteria is useful in terms of treatment strategies. *Id.* at 3-4. Significantly, the TMCWG states, "Although the criteria may be perceived as restrictive, we believe the use of these criteria will lead to the identification of more homogeneous groups of individuals for clinical studies." *Id.* at 4.

B. Petitioner's experts' opinions regarding the diagnosis

Dr. Agnes Jani-Acsadi opined that petitioner developed "a monophasic illness affecting his cervical and thoracic spinal cord consistent with a longitudinally extensive transverse myelitis lesion ("LETM") seronegative for aquaporin 4 antibodies." Pet. Ex. 18 at 8. She explained, "When the inflammatory lesion extends across more than three vertebral segments longitudinally, it is commonly referred to as 'longitudinally-extensive transverse myelitis.'" *Id.* at 3.

In his first report, Dr. Matthew Imperioli opined that petitioner "developed a monophonic illness affecting his cervical and thoracic spinal cord consistent with transverse myelitis within 4-6 weeks following the Tdap vaccination." Pet. Ex. 52 at 4. He stated that petitioner's "symptomology and documented neurological exams…is consistent with a predominantly thoracic myelopathy due to transverse myelitis." *Id.* He noted that petitioner was evaluated by neuroimmunologist, Dr. Mark Keegan at the Mayo Clinic, who "felt it was likely that [petitioner] had an inflammatory cause for the myelopathy." *Id.*

Additionally, Dr. Imperioli referenced the TMCWG's proposed inclusionary criteria and stated that "[petitioner] has none of the exclusion criteria" and "meets all the inclusion criteria except for the proposed time frame of progression to nadir of 4 hours to 21 days following the onset of symptoms." *Id.* at 5. Dr. Imperioli then argued that the TMCWG's proposed criteria were designed to be applied for recruitment of patients into research studies and therefore may be restrictive for clinical practice. *Id.*

He asserted, "Transverse myelitis is a heterogenous inflammatory disorder and not all patients may follow the same clinical course." *Id.* To support his assertion, Dr. Imperioli referenced an article by Kim et al., which provided a case report about a 51-year old woman whom developed progressive weakness in her upper and lower extremities over a four month period and was eventually diagnosed with neuromyelitis optica (NMO). Pet. Ex. 54 at 1.[14] The authors of this case report note that the patient's disease course did not meet the diagnostic criteria for idiopathic acute transverse myelitis, in which progression time to nadir from symptom onset is usually between four hours to three weeks. *Id.* at 2. The authors concluded

[14] Jun-Soon Kim et al., *A case of chronic progressive myelopathy,* 16(10) Multiple Sclerosis, 1255-1257 (2010). [Pet. Ex. 54].

that this case report demonstrates that patients with progressive myelitis over a period of several months can also have a limited form of NMO. *Id.*

Dr. Imperioli indicated that petitioner met the other inclusion criteria proposed by the TMCWG. Pet. Ex. 52 at 5. He stated that petitioner experienced sensory, motor and autonomic dysfunction attributable to the spinal cord, consistent with the TMCWG's inclusion criteria. *Id.* at 5. He also wrote, "In regards to the other inclusion criteria of transverse myelitis published by the TMCWG, [petitioner] had bilateral symptoms as well as a clearly defined sensory level on exam. MRI imaging excluded an extra-axial compressive lesion to account for his symptoms, i.e., there was no significant degenerative change of the spine or tumor to cause his myelopathy. There was evidence of inflammation within the spinal cord demonstrated by CSF pleocytosis." *Id.* Dr. Imperioli explained that petitioner had 8 cells in his first spinal fluid assessment and elevated CSF protein in his first spinal fluid assessment that occurred on November 26, 2012, as evidence of inflammation. *Id.* at 6; *see also* Pet. Ex. 11 at 186.

Dr. Imperioli stated that petitioner's MRIs "assist in narrowing the diagnosis to transverse myelitis." Pet. Ex. 52 at 6. He explained that the November 2, 2012 MRI of petitioner's cervical and thoracic spine revealed a T2 hyperintensity centrally in the spinal cord from C6-C7 interspace to the mid T3 level. *Id.*; *see also* Pet. Ex. 10 at 38-39. Dr. Imperioli stated, "This pattern is consistent with longitudinally extensive transverse myelitis as the lesion spans over 3 or more vertebral segments." *Id.* He also noted a "focal 2mm cyst area in the left side of the cord at T1." *Id.* Dr. Imperioli indicated that the petitioner's MRI in 2017 did not demonstrate any interval progression of the lesion over time, increased T2 hyperintensity within the cord and/or the development of contrast enhancement, which would be expected if the lesion was a neoplasm. *Id.* Importantly, he observed that the Mayo Clinic radiologist compared the 2017 MRI to petitioner's prior scans and reported a decrease in the T2 hyperintensity of the cord "which suggests interval improvement and is consistent with the expected evolution of imaging in transverse myelitis." *Id.*

Dr. Imperioli also stated that the entirety of the lesion is not consistent with that seen in a syringomyelia as there was "no dilation of the central spinal canal or significant expansion of the cord at the level of the lesion as would be expected." *Id.* Dr. Imperioli explained that syringomyelia are commonly associated with developmental disorders, such as Chiari malformations, or spinal cord trauma, spinal cord tumors and spinal cord infarction. Pet. Ex. 52 at 6. Dr. Imperioli suggested that the syrinx identified on petitioner's MRI could be caused by an inflammatory myelitis. *Id.* at 6. He cited an article by S. Ravaglia et al., which studied the development of syringomyelia in four patients with myelitis and found that the spinal cord cavitation developed in association with the myelitis and their "spatial coincidence suggested a causal relationship between the two conditions." Pet. Ex. 57 at 2.[15] The authors explained:

> Inflammatory diseases of the central nervous system (CNS) are characterized by the blood-brain barrier breakdown and abnormalities of the microvascular permeability, leading to edema. The accumulation of extracellular fluid increases the interstitial

[15] Sabrina Ravaglia et al., *Pathogenetic role of myelitis for syringomyelia*, 109 Clinical Neurology and Neurosurgery, 541-546 (2007). [Pet. Ex. 57].

> pressure in the spinal cord, leading to extension of the edema, and eventually, coalescence into syringomyelia.

Id. at 5. The authors concluded, "…our observations suggest that myelitis alone can induce the formation of syrinx as a result of intrinsic mechanisms, leading to accumulation of intra-spinal fluid." *Id.* at 6.

In Dr. Imperioli's second report, he expounded upon his initial opinion regarding petitioner's diagnosis after reviewing the petitioner's MRIs from the Mayo Clinic taken in 2017 and the respondent's experts' reports. Pet. Ex. 59. Dr. Imperioli stated that, "[Petitioner's] myelopathic symptoms reached a nadir at approximately 3-4 months based on the available records. *Id.* at 2. He conceded that the TMCWG's diagnostic criteria of acute transverse myelitis are the best criteria published to date and can apply to the majority of patients with acute transverse myelitis, but it is also common place to evaluate patients with a medical condition manifesting in an atypical manner from what is described in published literature. *Id.* at 2. But added that the TMCWG explained that proposed interval between symptom onset and maximal deficit is arbitrary. *Id.* at 2; *see also* Resp. Ex. A Tab-1 at 3. Dr. Imperioli referenced the article by Barreras et al., to demonstrate that some patients with inflammatory myelitis have a chronic progression of symptoms. Pet. Ex. 59 at 2.[16] Barreras et al. article reviewed 457 cases of patients with presumptive diagnosis of transverse myelitis to differentiate the clinical and paraclinical features of inflammatory myelopathy versus other causes of myelopathy (including vascular and spondylotic causes). *Id.* The authors classified 247 cases with inflammatory myelopathy. *Id.* They noted that those patients with inflammatory myelopathy had lesions that "more frequently occurred in the posterolateral spinal cord relative to other myelopathy groups and were located more often in the cervical and upper thoracic spinal cord (C1-T6)." *Id.* at 11. Additionally, Barreras et al. found that inflammatory myelitis presented more often with a subacute temporal profile, but that sixty-seven patients had a chronic progression of symptoms of 21 days or greater. Pet. Ex. 55 at 4. Further, of the 247 patients identified with inflammatory myelitis, 81% experienced sensory abnormality; 65% had demonstrated weakness during the neurologic examination; and 53% were found to be hyperreflexic. *Id.* at 12.

Dr. Imperioli stated that based on petitioner's records it was his opinion that, "causes of chronically progressive myelopathies, including hereditary myelopathy, spinal cord tumor, myelopathy due to a dural arteriovenous fistulas, and a chronic progressive form of multiple sclerosis were all sufficiently excluded." *Id.* at 2. He reiterated that petitioner's clinical presentation meets all the inclusionary criteria proposed by the TMCWG, except for the proposed time frame of progression and has none of the exclusion criteria. *Id.* at 3. Dr. Imperioli stated that the first lumbar puncture petitioner had on November 26, 2012 demonstrated evidence of inflammation as shown by elevated CSF protein and pleocytosis of 8 cells in the CSF, both were flagged as "high" on the lab report. Pet. Ex. 59 at 3; Pet. Ex. 11 at 186.

Dr. Imperioli again addressed the identification of the "small, focal cystic area in the left side of the cord at the T1 level," opining that the focal area is a small syrinx that was formed

[16] Barreras, P. et al., *Clinical biomarkers differentiate myelitis from vascular and other causes of myelopathy*, 90 Neurol. 12-21 (2018). [Pet. Ex. 55].

secondary to the myelitis and subsequent degenerative changes. Pet. Ex. 59 at 3. He explained that the "small, focal cystic area at the T1 level of the cord had a hyperintense signal different than the area of the myelitis, and has a similar signal intensity of the CSF. This cystic area does not appear to be communicating with the central canal as would typically be seen in syringomyelia." *Id. S*ignificantly, Dr. Imperioli observed that the small focal area was not identified on petitioner's initial MRI taken on October 26, 2012. *Id.*; Pet. Ex. 10 at 43. The neuroradiologist at the Mayo Clinic, Dr. David DeLone, reviewed petitioner's MRIs from October and November 2012, compared them to the MRI taken on March 18, 2013 and stated that a "small cystic appearing focus now present at C7-T1 was not clearly present on 10/26/2012." Pet. Ex. 7 at 33. Further, Dr. Imperioli wrote, "Dr. Brian Chin, the neurologist who read [petitioner's] 2017 MRI felt that the small focal cystic lesion was likely a syrinx but overall the cord lesion was suggestive of demyelination or an infectious inflammatory cause." Pet. Ex. 59 at 4. Dr. Imperioli stated that petitioner had no conditions commonly known to cause syringomyelia, such as Chiari malformations, Dandy-Walker cysts, basilar invaginations, spinal arachnoiditis, cord compression, spinal cord intramedullary hemorrhage and neoplasm, that could explain the appearance of the syrinx at the T1 level, other than it forming secondary to the myelitis that was demonstrated on petitioner's MRI. *Id.*

He concluded that petitioner developed a severe, monophasic immune mediated disease of his spinal cord consistent with transverse myelitis. Pet. Ex. 59 at 5.

C. Respondent's experts' opinions regarding petitioner's diagnosis

Dr. Donofrio opined that some of petitioner's treating physicians, "other than the neurologists," used the term "transverse-myelitis" without having a full understanding of the criterion used to establish a diagnosis of transverse myelitis. *Id.* at 7. He stated that neurologists are the most knowledgeable in the criteria for transverse myelitis and in differentiating the types of conditions that can cause a myelopathy (a generic term to describe all forms of spinal cord disease). *Id.* In his second report, he acknowledged that "the diagnosis of transverse myelitis was rendered in this case, even though petitioner's presentation did not meet the full criteria for transverse myelitis as outlined by the TMCWG in 2002." Resp. Ex. C at 4. Dr. Donofrio also agreed with Dr. Imperioli that the TMCWG's criteria was created for recruitment of patients into research studies and the criteria may be too restrictive for clinical practice. Resp. Ex. C at 3. However, he explained that he used the TMCWG's criteria to diagnose TM in his daily neurology practice and it is his opinion that "this is the practice of most of my colleagues." *Id.* at 1.

Dr. Donofrio summarized the TMCWG inclusion and exclusion criteria. Resp. Ex. A at 8-9. Dr. Donofrio stated that petitioner only met three of the six inclusionary TMCWG's criteria. *Id.* at 5. He explained:

> First, an extra-axial compressive etiology by neuro imaging was not excluded. Most physicians would consider a syrinx or intra-axial tumor as a compressive etiology. Second, inflammation within the spinal cord was never demonstrated by appreciable cerebral spinal fluid pleocytosis, an elevated IgG index or gadolinium enhancement.

> Third, the petitioner did not progress to the nadir of his illness within 4 hours to 21 days following the onset of symptoms.

Resp. Ex. A at 8. In his final report, Dr. Donofrio corrected himself, stating that "the imaging did exclude extra-axial compressive etiology." Resp. Ex. G at 2. Dr. Donofrio focused heavily on how petitioner's symptoms progressed, and, in his opinion, the lack of inflammation demonstrated in petitioner's CSF. Resp. Ex. A at 7-9; Resp. Ex. C at 3-6; Resp. Ex. G at 3.

In his first report, Dr. Donofrio stated that, "the petitioner was not found to be weak until four and half months after the Tdap vaccination or four months after the onset of the symptoms of pressure and burning over the abdomen." Resp. Ex. A at 7. Dr. Donofrio asserts that petitioner's first sign of weakness is on December 13, 2012. *Id.* However, in the same report, Dr. Donofrio acknowledged that when petitioner was evaluated by neurosurgeon, Dr. Darren Lovick on November 28, 2012, petitioner had difficulty with numbness in the legs, weak feet and gait instability. *Id.* at 7; Pet. Ex. 5 at 3.

In his second report, Dr. Donofrio explained that Dr. Keegan at the Mayo Clinic documented that petitioner's gait impairment began sometime after mid-October and "appears to have plateaued between December and January of this year." Resp. Ex. C at 3; Pet. Ex. 7 at 8. He stated that, "The timing of the progression of the disease in the petitioner is so far beyond the timing from the working group (3-4 months vs. 21 days), makes the diagnosis of TM untenable even if you consider the criteria too restrictive to be applied in all cases. This large difference in timing means the diagnosis of TM is unsupported." Resp. Ex. C at 4. Dr. Donofrio wrote, "Transverse myelitis is not a progressive illness beyond 3 weeks, yet the petitioner in this case clearly worsened over a much longer timeframe." Resp. Ex. G at 3. Dr. Donofrio noted that in May 2013, petitioner was utilizing forearm crutches and stated that, "The need for forearm crutches by May 2013 would be evidence for the progression of the thoracic myelopathy well into the year 2013, many months after the 21 days defined in the working group criteria for TM." Resp. Ex. G at 3.

Dr. Donofrio also asserted that petitioner did not have inflammation within the spinal cord demonstrated by laboratory testing, thus the petitioner would not meet the fifth criterion required by the TMCWG to make the diagnosis of TM. Resp. Ex. A at 9; Resp. Ex. G at 3. He stated that the petitioner did not have appreciable cerebral spinal fluid pleocytosis, an elevated IgG index or gadolinium enhancement of the spinal cord. *Id.* He agreed with Dr. Imperioli that the lack of gadolinium enhancement seen on petitioner's MRI may have been related to the timing of the imaging obtained. *Id.* Dr. Donofrio wrote that petitioner's spinal fluid findings showed 1 WBC and 1 RBC (normal results) on January 21, and the spinal fluid protein was elevated at 82 mg/dl (normal being less than 50). Resp. Ex. C at 5. He explained that "TM should cause an inflammatory reaction in the spinal fluid and one would expect a marked elevation of WBC to well above 5, the lower limit of normal." *Id.* While Dr. Donofrio did not address the petitioner's elevated WBC of 8 and elevated protein of 67 in his initial spinal fluid analysis, Dr. Donofrio asserted that petitioner did not have "convincing evidence for inflammation of the spinal cord by the spinal fluid analysis." Resp. Ex. G at 4.

Instead, Dr. Donofrio opined that petitioner "most likely had a syrinx (syringomyelia) or a low-grade tumor that began insidiously in August or September 2012 and progressed at least through 2013 when petitioner required forearm crutches. Resp. Ex. A at 10; Resp. Ex. C at 6. Dr. Donofrio stated that he had reviewed petitioner's MRIs from November 2, 2012 and that he agreed with the differential diagnosis rendered by the radiologist that included syringomyelia, a low-grade tumor or sequelae of transverse myelitis. Resp. Ex. C at 3. He also reviewed petitioner's MRIs performed on March 4, 2017 and stated that a focal 5 mm syrinx in the left aspect of the cord was noted at the T1 level. Resp. Ex. G at 1. Dr. Donofrio stated that the syrinx was present to his view in 2012 and 2017, but that "it does not appear that the syrinx is communicating with the ventricular system." *Id.* Dr. Donofrio cited to an article by Milhorat, which classified syringomyelia into communicating and noncommunicating syrinxes and possible causes of each, to refute Dr. Imperioli's position that transverse myelitis is a possible cause of syrinxes. Resp. Ex. F at 5;[17] Resp. Ex. C at 5.

He explained that petitioner's imaging revealed a syrinx, which is a "cavity within the spinal cord or lower part of the brain. They are most commonly localized between C2 and T9 of the spinal cord, the location of the myelopathy in the petitioner." *Id.* at 5. He stated that two months prior to receiving the vaccine, petitioner complained of urinary hesitancy and constipation, which would be consistent with the presence of a syrinx. Resp. Ex. G at 3. Dr. Donofrio also observed that petitioner's treating neurologist, Dr. Arkin, had suspected the diagnosis of syrinx and included in the differential diagnosis provided by the radiologist reading petitioner's MRI from November 2, 2012. *Id.* Dr. Donofrio stated, "Dr. Arkin assessed the [petitioner] four times and each time included a syrinx in his differential diagnosis." *Id.; see also* Pet. Ex. 3 at 10-11, 14-15, 20-22; Pet. Ex. 11 at 186, 192-94. However, he acknowledged that Dr. Mark Keegan, the neurologist at the Mayo Clinic, did not note the syrinx, but it was acknowledged by the radiologists. Resp. Ex. G at 2. He surmised that, "The Mayo Clinic neurologists may have not had the MRI scans reviewed by their colleagues in radiology." *Id.* However, the radiologist at the Mayo Clinic, Dr. David DeLone, did review petitioner's MRIs from October 2012 and November 2012. Pet. Ex. 7 at 31. Dr. DeLone observed, "The small cystic appearing focus now present at C7-T1 was not clearly present on 10/26/2012." *Id.* The radiologist, Dr. Kotsenas, who interpreted the MRI taken at the Mayo Clinic on March 18, 2013, observed, "At the C7-T1 interface there is a more focal-2 mm cystic appearing component in the left side of the cord." *Id.* Even after noting the cyst at C7-T1, Dr. Kotsenas' impression was, "Nonspecific T2 hyperintensity in the spinal cord C6-T3 could be secondary to NMO or post viral myelitis." *Id.*

Dr. Donofrio wrote that there are, "Many issues in this case refute the diagnosis of transverse myelitis." Resp. Ex. G at 3. He pointed to petitioner's symptoms of urinary and hesitancy beginning in June of 2012, two months before the Tdap vaccination, as evidence of pre-existing spinal cord disease. *Id.* at 3. Additionally, Dr. Donofrio noted that petitioner stated in his affidavit that he could walk an 18 hole golf course, carrying clubs, in mid-October which he did not find surprising as Dr. Arkin had noted normal strength and no weakness at the initial appointments. *Id.* He stated that, "Dr. Arkin rendered the diagnosis of possible syringomyelia at all four visits when he saw the petitioner," and that, "the petitioner had symptoms of spinal cord

[17] Thomas H. Milhorat, M.D., *Classification of Syringomyelia*, 8 Neurosurgery Focus (Mar. 2000). [Resp. Ex. F].

disease for 1-2 months before the August 7, 2012 vaccination." *Id.* at 4. He concluded that it was his opinion that petitioner did not have transverse myelitis. *Id.*

D. Petitioner has demonstrated that he had suffered transverse myelitis

After reviewing petitioner's medical records, the experts' opinions and medica literature, I have determined that petitioner has provided preponderant evidence that he suffered transverse myelitis.

1. Diagnosis of Treating Providers

At his first appointment with neurologist Dr. Arkin on October 30, 2012, Dr. Arkin considers the possibility of petitioner having a syrinx or "inflammatory/autoimmune or demyelinating multiple sclerosis." Pet. Ex. 10 at 25. On November 2, 2012, petitioner had a second MRI of his cervical and thoracic spine. *Id.* at 37. The radiologist's impression was, "Intramedullary central T2 central signal extending from the C6-C7 to T3-T4 level as well as a left lateral cystic appearing lesion at the T1 level. Primary differential consideration includes a syringomyelia, sequelae of prior inflammation (transverse myelitis)." *Id.* at 38. After review of petitioner's second MRI, taken on November 2, 2012, Dr. Arkin observed a "tiny 5 x 3 mm T2 lesion just laterally within the cord which looks cystic," and stated that he suspected a syringomyelia, but that it "could also be transverse myelitis associated with collagen vascular disease or other type of autoimmune pathology." *Id.* at 23. On November 26, 2012, Dr. Arkin noted that petitioner had "subacute and progressive onset of circumferential paresthesias and dysesthesias involving the abdomen with a sense of weakness in the legs," and that "the suggestion is inflammation versus syrinx given the midline distribution. I think he probably has syringomyelia." Pet. Ex. 3 at 17. After petitioner underwent an arteriogram which ruled out a dural arteriovenous fistula, neurosurgeon, Dr. Darren Lovick opined that petitioner could have an inflammatory condition like, "transverse myelitis, sarcoidosis and demyelinating processes…." Pet. Ex. 5 at 5. He recommended petitioner begin prednisone. *Id.* As of January 10, 2013, despite the negative arteriogram petitioner's differential diagnosis continued to be, "fistula versus sarcoidosis versus transverse myelitis." Pet. Ex. 3 at 13.

On March 18, 2013, petitioner sought a tertiary care second opinion from the Mayo Clinic. Pet. Ex. 7 at 19. He had repeat cervical and thoracic spine MRIs, which again demonstrated the T2 hyperintensity in the spinal cord at C6-T3 and a small "cystic appearing component in the left side of the cord." Pet. Ex. 7 at 31. The radiologist interpreting the March 18, 2013 MRI stated that the observed hyperintensity "could represent a demyelinating process such as neuromyelitis Optica or post viral myelitis. The absence of enhancement would be atypical for a neoplastic process or sarcoidosis." *Id.* Dr. David LeLone compared petitioner's MRIs taken on October 26th and November 2, 2012 and observed that, "The small cystic appearing focus now present at C7-T1 was not clearly present on 10/26/2012." *Id.* On March 20, 2013, Dr. Joseph Matsumoto reviewed petitioner's neuroimaging from October 26, 2012 and compared it to the MRI taken on March 18, 2013. *Id.* at 14. He wrote, "[Petitioner] had a repeat MRI of the spine. This showed a multi-segment T2 change consistent with a long area of intrinsic myelopathy…There was no evidence of AVM or AV fistula according to Dr. Kotsenas….To me, on the films, the scan of October 26, 2012, looks if anything a bit worse than

the one we have today. The area of T2 signal abnormality looks a little bit wider within the cord than what we see today." *Id.* Dr. Matsumoto also noted that Dr. Kotsenas also appeared to rule out a tumor and decided to have petitioner meet with a demyelinating expert, Dr. Mark Keegan. *Id.* Dr. Keegan evaluated petitioner on March 21, 2013. *Id.* at 11. Dr. Keegan stated that he "personally reviewed the neuroimaging" and after taking petitioner's history and a physical exam, stated, "It seems likely that [petitioner] had an inflammatory cause for the myelopathy….If all the serological evaluations came back negative, this could remain, even if this is an autoimmune inflammatory cause, a monophasic process." *Id.* Dr. Keegan diagnosed petitioner with "Myelopathy, probable inflammatory cause." *Id.*

On March 28, 2013, when petitioner sought an evaluation for his rehabilitation needs, Dr. Brad Steinle diagnosed petitioner with transverse myelitis with paraparesis, neuropathic pain, neurogenic bladder and bowel and recommended physical therapy and prescribed Nuvigil.[18] Pet. Ex. 10 at 11. Dr. Steinle maintained petitioner's diagnosis of transverse myelitis at a follow-up appointment on August 6, 2013 and on February 19, 2015. Pet. Ex. 14 at 5-7. In February 2017, at an appointment with neurologist Dr. James McDonald for an evaluation of intermittent prickly dysesthesias in petitioner's upper arms, Dr. James McDonald noted petitioner's history of transverse myelitis "beginning in 2012" and recommended a repeat MRI. Pet. Ex. 51 at 1. Dr. McDonald reviewed the March 4, 2017 MRI and compared it to the MRI performed in November 2012. *Id.* at 8. Dr. McDonald observed that the overall T2 signal was decreased from the study performed in November 2012 and he also noted a "multifocal area of T2 hyperintensity likely a syrinx (at the level of T1)," but concluded, "MRI of the cervical spine without and with contrast showed an overall decreased T2 signal compared to the last study most likely suggesting either a remote infectious/inflammatory process or demyelination." *Id.* at 8. Dr. McDonald also stated that he reviewed petitioner's records from the Mayo Clinic and that, "It was felt that he had idiopathic transverse myelitis." *Id.*

Petitioner's medical records and treating neurologists establish the diagnosis of transverse myelitis and they rule out possible other causes, such as sarcoidosis, arteriovenous fistula and a neoplastic process. Dr. Donofrio puts significant weight on the presence of a 5x3 mm syrinx at the T1 level, recognized on petitioner's MRIs from November 2012, March 2013 and March 2017, as the cause for petitioner's symptoms and disease. However, he also noted that "a radiologist lists a differential diagnosis of potential disorders, *leaving the final decision to the clinician caring for the patient*." Resp. Ex. G at 3 (emphasis added). Importantly, petitioner's interpreting radiologists and treating neurologists acknowledged the syrinx, but concluded that petitioner had suffered an inflammatory myelopathy that was consistent with transverse myelitis. For example, Dr. Mark Keegan explained that he had reviewed petitioner's neuroimaging and came to the conclusion that petitioner had a myelopathy with a probable inflammatory cause. *See* Pet. Ex. 7 at 10-11. Further, Dr. Matusumoto acknowledged the identification of a "small cystic-appearing focus…at C7-T1," but his impression was consistent with Dr. Keegan's-that petitioner had "an inflammatory myelopathy," and referred to petitioner's condition as "a monophasic illness such as transverse myelitis." Pet. Ex. 7 at 7. Petitioner's neurologist Dr. McDonald did the same, he recognized the existence of a syrinx at the level of T1, but concluded that the "persistent abnormal cord signal at the cervicothoracic junction with overall decrease T2

[18] Nuvigil (armodafinil) is used to treat excessive sleepiness caused by narcolepsy or shift work sleep disorder.

signal," was suggestive of a remote inflammatory process or demyelization. Pet. Ex. 51 at 8. The

Finally, the Ravalia et al. article, cited by Dr. Imperioli, suggests that the identified syrinx developed in conjunction with petitioner's transverse myelitis. The article reviews four cases of individuals with spinal cord inflammation and syringomyelia and found that "syrinxes associated with myelitis shared some common features: they developed during the acute phase of myelitis and disappeared after steroids, were all non-communicating cavitations involving the central canal and occurred in the same spinal segment affected by myelitis." Pet. Ex. 57 at 1. The authors concluded that the myelitis alone can induce the formation of a syrinx as a result of intrinsic mechanisms, leading to accumulation of intra-spinal fluid. *Id.* at 6. They also stated that the, "CSF-flow imbalance can accelerate the development of syringomyelia, especially in myelitis characterized by discrete focal lesions." *Id.* In petitioner's case, he had a discrete focal lesion identified on his MRI from C6-7to T3-4 and the small syrinx was identified in the same area of his spine at T1. Pet. Ex. 51 at 20. Additionally, both experts classified the syrinx as non-communicating. Resp. Ex. G at 1; Pet. Ex. 59 at 3. Even though petitioner's syrinx did not appear to have resolved with steroid treatment, he was treated with a lower dose and shorter course of oral prednisone than the patients discussed in the Ravalia et al. article. Although Dr,.Arkin leaned toward a diagnosis of syringomyelia, the later treating neurologists and radiologist, particularly those at the Mayo Clinic, made a diagnosis of an inflammatory myelopathy such as transverse myelitis.

2. Application of TMCWG's criteria for idiopathic acute transverse myelitis

The main contention between the parties is whether the proposed inclusionary criteria of acute idiopathic transverse myelitis by the TMCWG should be applied in this case to support the diagnosis of transverse myelitis. Pet. Brief at 18-19. Resp. Brief at 17.

Dr. Imperioli acknowledge the existence of the proposed diagnostic criteria by the TMCWG and stated that petitioner had met all of the inclusionary criteria, except for the proposed frame of progression to nadir of 4 hours to 21 days following the onset of symptoms and he had none of the exclusionary criteria. Pet. Ex. 52 at 5. However, Dr. Imperioli argued that the TMCWG proposed criteria was designed for the recruitment of patients into clinical research studies and therefore may be too restrictive in clinical practice. Pet. Ex. 59 at 2. He observed that even the authors acknowledged that the "interval between symptom onset and maximal deficit is arbitrary," and that "they remain committed to distinguishing ATM from a rapidly evolving vascular myelopathy or a slowly progressive or stuttering myelopathy, spinal cord tumor, myelopathy due to a dural arteriovenous fistula, and a chronic form of MS." Resp. Ex. A, Tab 1 at 3. Dr. Imperioli stated that, "The proposed criteria applies for the majority of patients with acute transverse myelitis, but in clinical practice, it is common to evaluate a patient with a medical condition manifesting in an atypical manner from what is described in text books and other published literature." Pet. Ex. 59 at 2.

Dr. Donofrio agreed with Dr. Imperioli that the proposed criteria developed by the TMCWG was developed for recruitment of patients into research studies and that the criteria

may be too restrictive for clinical practice. Resp. Ex. C at 3. He continued, however, and stated that, "…they are the best inclusion and exclusion criteria thus far," and that he utilizes the TMCWG's criteria in his daily practice to diagnose TM and believes most of his colleagues do as well. *Id.*; Resp. Ex. G at 1. Dr. Donofrio initially opined that petitioner only met three out of the six inclusionary criteria proposed by the TMCWG in his first report, but modified his opinion by his last report, noting that extra-axial compressive etiology was excluded. Resp. Ex. A at 5; Resp. Ex. G at 2,4. Specifically, Dr. Donofrio stated that petitioner did not meet the proposed inclusionary criteria for transverse myelitis because petitioner did not have a monophasic illness which reached its nadir within 21 days from the onset of symptoms and that he did not have convincing inflammation of the spinal cord by spinal fluid analysis. Resp. Ex. G at 4.

Dr. Imperioli explained that petitioner's symptoms reached a nadir at approximately 3-4 months based on the records. Pet. Ex. 59 at 2. Petitioner stated that he first began experiencing his noticeable symptoms of abdominal pain and difficulty urinating in late September. Pet. Ex. 49 at ¶ 1. He stated that he began to experience pain that was like a band with pressure around his abdomen that built up over time, resulting in him seeking medical assistance on October 18, 2012. *Id.* at ¶ 2. Petitioner explained that by the time he was able to see his primary care physician, Dr. Stiles, he was feeling weak and was unable to walk significant distances. *Id.* at ¶ 3. The "Indication" field on petitioner's October 26, 2012 MRI states, "Mid abdominal pain and tightness. Difficulty walking. Tingling in the bilateral upper extremities. PAIN." Pet. Ex. 10 at 42. This record supports petitioner's statement that he began to have difficulty walking by late October 2012. When petitioner saw Dr. Lovick on November 28, 2012, it was recorded that petitioner had difficulty with his gait and difficulty with numbness in his legs and weakness in his feet. Pet. Ex. 5 at 3. On December 26, 2012, petitioner returned to Dr. Lovick following the arteriogram, which was negative for "any type of arteriovenous malformation," and it was noted that petitioner "is not getting any worse" and he was "clinically stable." *Id.* at 5. When petitioner presented at the Mayo Clinic for an evaluation, he told Dr. Keegan that "there was no clear worsening recently," and Dr. Keegan concluded that petitioner's symptoms of gait impairment plateaued between December and January [of 2013]. Pet. Ex. 7 at 8.

Dr. Donofrio asserted that petitioner's symptoms progressed "at least through 2013 when the petitioner required forearm crutches." Resp. Ex. C at 6. In his opinion, the petitioner did not have a monophasic illness and that petitioner's nadir was not reached for 3-4 months after the onset of his symptoms of pressure and burning over the abdomen. *Id.* However, petitioner utilizing the forearm crutches in 2013 was the result of him following Dr. Keegan's recommended course of action and not evidence that his disease had progressed. *See* Pet. Ex. 7 at 11 (recommending petitioner utilize a gait aid for fall prevention).

While Dr. Imperioli conceded that petitioner's disease progression was longer than the TMCWG's proposed criteria, he cited to medical literature which supports a longer progression of symptoms than included in the TMCWG's criteria. The Barreras article found that 67 patients out of 247 with inflammatory myelopathy had a progression of symptoms of 21 days or greater. Pet. Ex. 55 at 5; Pet. Ex. 59 at 2. The authors explained that a hyperacute presentation of symptoms was suggestive of a spinal cord ischemic stroke and a chronic evolution was suggestive of a vascular lesion, such as an arteriovenous fistulas or arteriovenous malformation. Pet. Ex. 55 at 5. In petitioner's case, the arteriovenous fistulas and arteriovenous malformation

were excluded by his treating physicians. *See* Pet. Ex. 5 at 5 (Dr. Lovick noting petitioner's arteriogram was negative for a dural AV fistula or any type of arteriovenous malformation.). Thus, while not the classic presentation of transverse myelitis as defined by the TMCWG, the symptom development and imaging in petitioner's case appeared more likely than not to be the result of an inflammatory process rather than a dural fistula which was ruled out or the syrinx, which may well have been secondary to the initiating myelitis. Additionally, petitioner's symptoms did not appear to progress after December 2012, when Dr. Lovick noted that petitioner was "clinically stable," and petitioner's symptoms had not progressed since the prior visit on November 28, 2012. *See* Pet. Ex. 5 at 5. They appeared to have plateaued by the time he went to the Mayo Clinic and Dr. Kegan's recommendation of using a walker was for fall protection not for progressing symptoms.

The TMCWG's third criterion is that inflammation is demonstrated in the spinal cord by CSF pleocytosis *or* elevated IgG index *or* gadolinium enhancement. Resp. Ex. A, Tab 1 at 2. The criterion specifically states, "If none of the inflammatory criteria is met at symptom onset, repeat MRI and lumbar puncture evaluation between 2-7 days following symptom onset to meet criteria." *Id.* Petitioner's first MRI, on October 26, 2012, was done without the administration of intravenous contrast. Pet. Ex. 10 at 42. His second MRI was done with enhancement and found a "redemonstration of central T2 hyperintensity extending from C6-C7 to the T3-T4 level," and, "there was no associated enhancement." *Id.* at 38. Both Dr. Imperioli and Dr. Donofrio acknowledged that the lack of gadolinium enhancement on petitioner's second MRI may have been due to the timing of the imaging obtained. *See* Pet. Ex. 52 at 6; Resp. Ex. C at 4.

Dr. Donofrio, however, argued that petitioner's spinal fluid findings did not demonstrate pleocytosis, as petitioner's spinal fluid showed 1 white blood cell and his spinal fluid protein elevation of 82 mg/dl was non-specific and would not support a diagnosis of TM without an elevation of WBC count. Resp. Ex. C at 5. However, Dr. Donofrio's reference is only to petitioner's second lumbar puncture, taken on January 21, 2013. Petitioner's first lumbar puncture, taken on November 26, 2012, revealed a slightly elevated protein level of 67 in his CSF and a white blood cell count of 8/ul. Pet. Ex. 10 at 20; Pet. Ex. 11 at 186. The lab report indicated that petitioner's WBC count was "high." Pet. Ex. 11 at 186. Dr. Imperioli stated that evidence of inflammation within the spinal cord demonstrated by CSF pleocytosis is usually considered an elevation of greater than 5 leukocytes/ul. Pet. Ex. 59 at 3. Additionally, the article by Barreras found that 43% of patients who met the criteria for inflammatory myelitis did not exhibit pleocytosis and 46% of patients had elevated protein (>45 mg/dl) in the CSF. Pet. Ex. 55 at 6. The record demonstrates that petitioner had mild CSF pleocytosis and thus would meet the TMCWG's third inclusionary criterion.

The record overall supports a finding that petitioner suffered from transverse myelitis. Petitioner's treaters were able to rule out possible alternative causes, such as an arteriovenous fistula, neoplastic process, or sarcoidosis. Further, the identification of a small syrinx on petitioner's MRI did not change the impression of his treating physicians, particularly those at the Mayo Clinic, that petitioner had an inflammatory myelopathy categorized as transverse myelitis after the comprehensive evaluation at the Mayo Clinic. Finally, even if petitioner did not meet all of the proposed diagnostic criteria for idiopathic transverse myelitis, experts from both parties, along with the authors of the proposed Transverse Myelitis Working Group criteria,

agree that there are limitations to the criteria and the purpose of these restrictive criteria was to "lead to the identification of more homogenous groups of individuals for clinical studies." *See* Resp. Ex. A, Tab 1 at 4. Therefore, based on the clinical judgment of his physicians, particularly those at the Mayo Clinic who reviewed his history, all of his films and did a comprehensive evaluation, I find that petitioner presented preponderant evidence that the petitioner's appropriate diagnosis is transverse myelitis.

V. *Althen* Prongs

In order to demonstrate entitlement for an "off-table" injury, petitioner must demonstrate all three elements established by the Federal Circuit in *Althen.* 418 F. 3d 1274, 1278. The petitioner must establish, by preponderant evidence: (1) a medical theory causally connecting the vaccine and her injury; (2) a logical sequence of cause and effect showing that the vaccine was the reason for his injury; and (3) a showing of proximate temporal relationship between the vaccine and his injury. 418 F.3d at 1278.

In this case, the petitioner's most difficult problem in proving causation is regarding the acceptable timing criteria of *Althen* prong three. The third prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe, which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation. *De Bazan v. Sec'y of Health & Human Servs.,* 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must also coincide with the theory of how the relevant vaccine can cause an injury. *Id.*; *see also Shapiro v. Sec'y of Health & Human Servs.,* 101 Fed. Cl. 532, 542 (2011), *recons. denied after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem*., 2013 WL 1896173 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Human Servs.,* No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for review denied* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

Dr. Imperioli opined that there are several mechanisms by which antigens in vaccines can induce transverse myelitis. Pet. Ex. 52 at 6. Dr. Imperioli referenced an article by Agmon-Levin et al.[19] which reviewed reported cases of transverse myelitis following vaccination, to support his opinion that vaccines may induce transverse myelitis through molecular mimicry, epitope spreading or bystander activation. *Id.* at 6-7. Dr. Imperioli also noted that the Agmon-Levin et al. article identified 37 cases of post-vaccination transverse myelitis, which included 5 patients with transverse myelitis following the DTP or DT vaccines and 1 following a pertussis vaccination. Pet. Ex. 59 at 4; Pet. Ex. 39 at 14. Petitioner's causation theory offered by Dr. Imperioli includes molecular mimicry, which has been generally accepted as a reputable and reliable scientific theory for explaining the pathophysiology of certain immune-mediated conditions. *See White v. Sec'y of Health & Human Servs.,* No. 15-1521V, 2019 WL 7563239 (Fed. Cl. Spec. Mstr. Dec. 19, 2019); *Forrest v. Sec'y of Health & Human Servs.,* No. 14-1046V, 2019 WL 925495 (Fed. Cl. Spec. Mstr. Jan. 28, 2019); *Hargrove v. Sec'y of Health & Human Servs.*, No. 05-0694V, 2009 WL 1220986, at *38 (Fed. Cl. Spec. Mstr. Apr. 14, 2009).

[19] Agmon-Levin, N. et al, *Transverse Myelitis and vaccines: a multi-analysis*, 18 Lupus 1198-1204 (2009). [Pet. Ex. 39].

As noted above, the petitioner is also required to show how the medically acceptable timeframe for the onset of symptoms coincides with the theory of causation. The timing of the onset of the petitioner's transverse myelitis is a challenging problem, and in this case, is fatal to the claim.

Dr. Imperioli stated that petitioner's symptoms began 4-6 weeks after the vaccination, which is a medically acceptable timeframe for transverse myelitis symptom onset. Pet. Ex. 52 at 7. Dr. Donofrio stated that if petitioner's symptom began in late September or early October 2012, the symptoms are too delayed to attribute them to the onset of symptoms to the vaccination of August 7, 2012. Resp. Ex. C at 4. He stated, "One would expect an immune response to occur within 7-10 days after a vaccination, if implying an immune response modulated by molecular mimicry, epitope spreading and induced autoimmunity from infectious agents or vaccination." *Id.* Dr. Fujinami also noted that if the Tdap vaccine contained cross-reacting epitopes with myelin following immunization, the clinical manifestations would likely occur within 2 weeks following vaccination, which is a very different time course than that experienced by the petitioner. Resp. Ex. H at 4.

Petitioner received the Tdap vaccination on August 7, 2012. Pet. Ex. 1 at 1. On October 18, 2012, he presented to the University of Kansas Hospital urgent care, reporting mild discomfort in the lower abdomen and urinary symptoms for "the last several days." Pet. Ex. 6 at 5. When he sought treatment from his primary care physician on October 23, 2012, he reported, "a week to 10-day history of upper abdominal discomfort," and increasing problems of urinary hesitancy. Pet. Ex. 10 at 5. Thus, based on the history provided at petitioner's early appointments in October 2012, and taking the version most favorable to the petitioner, the onset would have been on or about October 13, 2012, or 67 days post-vaccination

At the Mayo Clinic, on March 18, 2013, or five months after the earlier reported date of onset, petitioner told Dr. Matsumoto that, "In September he began to feel a tightness in his abdomen," and he experienced a sense of burning. Pet. Ex. 7 at 17. Then on March 21, 2013, petitioner explained to Dr. Keegan that, "In about September or perhaps earlier, he developed a numb sensation and tightness and burning sensation in his abdomen." *Id.* at 10. However, in petitioner's affidavit, executed on November 29, 2017, petitioner stated, "….I did not have any noticeable symptoms until late September to early October." Pet. Ex. 49 at ¶ 2. He explained that he was having trouble urinating, along with the abdominal pain. *Id.* He also described that his pain felt like a band, with pressure across my abdomen. *Id.*

In his motion, petitioner argues that a medically appropriate timeframe of the onset of transverse myelitis following the Tdap vaccination is 2-63 days. Pet. Mot. at 22. Petitioner references the Agmon-Levin article to support a temporal association as long as three months between vaccination and the onset of transverse myelitis. Pet. Ex. 39 at 1. It is important to note that of the 37 cases in the Agmon-Levin study, only nine of the cases occurred more than 21 days post vaccination and three of those were at implausibly long intervals of four, six and nine years post vaccination. *Id.* at 4. The three-month timeframe from vaccination to onset was in relation to one Hepatitis B vaccination. *Id.* at 4. *Id.* at 4. Of the DTP, DT or pertussis[20]

[20] The Agmon-Levin study did not report any cases involving TDaP vaccination, but I have considered all of the vaccines containing component antigens of the TDaP vaccine.

vaccinations, the intervals from injection to onset of symptoms were between three and seventeen days. *Id.*

Dr. Donofrio's opinion that symptom onset would have to occur between seven and ten days post-vaccination is considerably shorter than accepted time periods in other vaccine cases, however, the interval of 67 days for symptom onset falls outside a medically accepted temporal association with vaccine causation. Even if it is assumed that petitioner's symptoms began in late September, the interval would be approximately 54 days post-vaccination, and would l have begun outside the 4-6 week (28-42 day) window suggested by Dr. Imperioli. At petitioner's first medical appointment on October 18, 2012, he reported "several days" of abdominal pain, thus putting onset of symptoms in mid- October. His second estimate of the date of onset given on October 23, 2012, as seven to ten days is also consistent with a mid-October onset. Petitioner's statements in his supplemental affidavit rendered on November 29, 2017, places the onset of pain in his bladder in "late September to early October." Taking the latest time for medically acceptable temporal association to the vaccine as proposed by Dr. Imperioli of 6 weeks or 42 days post-vaccination, the symptom onset would have had to occur by September 18, 2012. There was no convincing evidence that the symptoms began on or before that date. The medical records and petitioner's statements persuasively put the onset of petitioner's symptoms in mid-October but no sooner than the end of September, thus falling outside of Dr. Imperioli's suggested timeframe for a medically acceptable temporal association with the Tdap vaccination.

Additionally, for petitioner's claim an earlier onset for his condition makes transverse myelitis less likely, in that transverse myelitis usually peaks within 21 days and, as observed at the Mayo Clinic, his condition did not plateau until late December 2012 or early January 2013. Even with a mid-October date of onset, the timing from onset to nadir in this case is unusual but not impossible for transverse myelitis as demonstrated by some of the medical literature submitted. However, if the onset of petitioner's symptoms began in mid-September, as suggested by petitioner to Mayo Clinic physicians, it makes the progression of his symptoms less likely to fit transverse myelitis.

Special masters have found that a petitioner was entitled to causation where onset occurred up to two months, or 56 days following the flu vaccination. *Spayde v. Sec'y of Health & Human Servs.*, No. 16-1499V, 2021 WL 686682 (Fed. Cl. Spec. Mstr. Jan. 27, 2021) (accepting a 56-day onset for GBS following flu vaccination) (citing to *Barone v. Sec'y of Health & Human Servs.*, No. 11-707V, 2014 WL 6834557, at *13 (Fed. Cl. Spec. Mstr. Nov. 12, 2014) (finding a six-week or 42-day interval from vaccination to onset of petitioner's GBS). However, these are cases involving the influenza vaccine and Guillain-Barré syndrome, and not the occurrence of transverse myelitis following a Tdap vaccine. Further, other compensated cases where petitioners suffered from transverse myelitis following the Tdap vaccination had onset of symptoms much closer in time to the administration of the vaccine. *See e.g. Roberts v. Sec'y of Health & Human Servs.*, No. 09-427V, 2013 WL 5314698 (onset of symptoms began four weeks after Tdap vaccination); *Helman v. Sec'y of Health & Human Servs.*, No. 10-813V, 2012 WL 1607142, at *3 (Fed. Cl. Spec. Mstr. Apr. 5, 2012) (finding onset of symptoms occurring three weeks after Tdap vaccination); *Raymo v. Sec'y of Health & Human Servs.,* NO. 11-0654V, 2014 WL 1092274, at *19 (Fed. Cl. Spec. Mstr. Feb. 24, 2014) (finding a three to four-day onset of

transverse myelitis following administration of Tdap vaccine to medically appropriate temporal relationship).

The Agmon-Levin article, which examined only 37 cases of transverse myelitis after vaccination cannot be considered absolutely definitive in establishing an outer time line for vaccine causation, it is the best evidence presented by the petitioner, and it reported onset of transverse myelitis at most 17 days post-vaccination for DPT, DT or pertussis. Further, Vaccine Program cases involving the Tdap vaccine have found vaccine causation where onset has occurred 28 days post-vaccination. I do not consider four weeks or 28 days for onset to be a hard line for establishing a medically appropriate timeframe, however, 54 to 67 days post-vaccination for symptom onset is beyond the time period for which a medically acceptable causal timeframe can be established based on current medical understanding and the evidence submitted in this case. Therefore, I find that the petitioner has not established by preponderant evidence that his symptoms began within a medically acceptable timeframe to satisfy *Althen* prong three.

VI. Conclusion

The petitioner has not carried his burden of proof, and therefore is not entitled to an award of compensation. In absence of a motion for review filed pursuant RCFC Appendix B, the Clerk of the Court shall enter JUDGMENT in accordance with the terms of this decision.

IT IS SO ORDERED.

s/Thomas L. Gowen
Thomas L. Gowen
Special Master